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CROWN-ROT OF FRUIT TREES: HISTOLOGICAL STUDIES

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INTRODUCTION

The term "crown-rot" is used to designate a bark disease of fruit trees (chiefly apple—*Pyrus malus* and pear—*Pyrus communis*), occurring in most of the tree-growing portions of the United States. The affected bark eventually dies in various sized patches, and the surrounding living tissues produce callus, which thus separates the living tissues from the dead. This disease most characteristically affects the bark of the lower trunk and that of the adjoining portions of the upper roots. The location of affected patches of bark seems to depend very largely upon the interrelation of growth and weather conditions; in some cases the disease involves chiefly the upper roots, while in other cases it occurs most frequently at the base of the trunk. An affected patch of bark that dies to the wood decays more or less rapidly, depending upon its distance from the ground or other sources of moisture. The wood exposed by the decayed bark is usually discolored at its surface but may be alive within and active in the conduction of water. When the crown-rotted patch extends around three fourths or more of the trunk, the downward current of elaborated food in the bark is interfered with to such an extent as to permit much less than the normal amount of radial growth in the roots. The enfeebled roots thus absorb less soil solution, and therefore smaller leaves are formed. The wood under the wound dies in time, and thus the water-conducting tissues are reduced. Such trees usually die in a few years unless radial growth produces much new wood about the wound in the meantime. In some instances the bark dies entirely around the base of the trunk, and in many cases of this sort the width of the dead girdle determines the length of time such a tree will live.

Crown-rot has an important economic bearing upon the fruit industry of this country, owing to the fact that it involves the lives of trees and is therefore much more serious than fruit- and leaf-spotting diseases which after all are essentially matters of a season.

Crown-rot and related bark diseases have been investigated intermittently during many years; in the early days of phytopathology considerable attention was given to these diseases and much valuable information was accumulated. The subsequent enormous development of mycology, in its relation to the diseases of plants in general, has eclipsed and supplanted the interest formerly centered on bark diseases, apparently because the mycological phases afforded more tangible subjects for investigation. Most of the more modern attempts at the study of crown-rot have been of a preliminary nature and have led only to hazy or ill-founded conclusions.

Some of the apparently new ideas that occurred to me during the earlier part of this investigation were published in 1909,[1] while others were stored away, embodied in the form of notes and photographs to be used later. Continued search of the older literature of botany and forestry for observations upon bark diseases, as well as with reference to the question of radial growth,[2] resulted in gradually placing one after another of my supposed new ideas in the category of confirmatory observations and conclusions.

The Literature.—The literature accumulated on crown-rot and related subjects during the past six years has become very voluminous, and to attempt a review seems rather discouraging. Most of the important papers on radial growth, and on certain of the factors determining its distribution, were reviewed some time ago in the last cited paper. Many of the more general papers on this disease and some of those dealing with the cause of the trouble were discussed in my two former papers on crown-rot. There are still too many abstracts of such papers on hand to be fully utilized in this connection, and therefore only a few of the most pertinent ones will be mentioned later in the discussion of my results.

The Causes of Crown-Rot.—The common orchard bark-fungi are evidently the causes of the rotting of the bark in crown-rotted trees, but the cause of the initial injuries that led to the death of this bark has not been experimentally determined, although some work upon this problem has been done on citrus trees in Florida. Field observations in the north, together with a few experiments, have shown that the manner and timeliness of radial growth, pruning, and the occurrence of droughts or low temperatures are closely related to the development of crown-rot there; and similar investigations in Florida are revealing the prevalence of comparable adverse environmental conditions, preceding the occurrence of foot-rot on citrus trees. In the latter case it was also noticed that the leaf-surface area of a tree exposed to sufficient light for active photosynthesis, when compared to the surface area of bark on the branchless portion of the trunk, is often rather small in trees which appear especially subject to this disease; that is, it is suggested that a scarcity of elaborated food coursing downward in the bark of the trunk below the branches may also have a causal relation to the occurrence of crown-rot.

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[1] Crown-rot, Arsenical Poisoning and Winter Injury, N. Y. Agr. Exp. Sta. Tech. Bull. **12**: 369. 1909.

[2] The Periodicity and Distribution of Radial Growth in Trees and Their Relation to the Development of "Annual" Rings, Trans. Wisc. Acad. Sci. Arts and Letters **18**: 1. 1915.

Methods of Work.—It was found possible to cut usable sections from material of small diameter embedded in celloidin without the previous use of softening agents. The series used in this study was obtained chiefly in the manner described and from material collected and fixed in spring and summer of 1912. The citrus material was collected later in Florida and similarly treated.

Flemming's strong solution and Carnoy's mixture were used almost exclusively for fixation. The former gave more satisfactory material for staining, but the latter was more largely used because of its more rapid penetration. Flemming's triple stain and Heidenhain's iron-alum haematoxylin stain were most frequently used. The triple stain was found to yield much quicker and more satisfactory results when used after a mordant such as iron-alum, but for use in making photographs iron-alum haematoxylin proved more desirable than Flemming's. The same was later found to be the case in sections from foot-rot material of citrus trees.

The Early Stages of the Disease.—The first visible stages of crown-rot consist of discolored and often ruptured tissues variously distributed in streaks and patches in the bark. In cross-section the injured patches are often arranged more or less concentrically about the wood cylinder, although they are usually most severe on one side of the stem. In the mildest forms of the disease the medullary rays of the inner phloem and groups of parenchyma cells about the sclerenchyma strands and inner cortex are affected, although at times only the one or the other of these tissues is involved. In more severe cases much of the phloem and practically all the cambium may be injured.

The severity and course of the disease following these evident beginnings depends mainly upon the relative extent and number of the injured or dead patches, upon the weather of the ensuing growing season, and possibly upon the relative abundance of wound fungi. In many of the milder cases, the injured and collapsed tissues are merely more or less compressed by the subsequent growth of the surrounding live parenchyma cells, and in late summer only the presence of irregular formless dead masses among the living tissues of the bark tells the story of the former trouble. In the more severe cases, however, in which in addition to the medullary rays of the phloem, the inner portions of the cortex and perhaps most of the cork cambium have been much injured, the results are likely to be more serious. In these cases, as in the milder ones, the resumption of growth by the surrounding live parenchyma results in the compression of the dead and dying tissues; but since the dead patches are numerous, relatively large, and close together, the intervening live parenchyma and ray-cells are insufficiently supplied with water and nutrients and therefore cannot survive the drying weather of late spring and early summer. During the latter part of this process a new cork cambium is developed inside the dying cortical parenchyma, resulting afterward in a rough, scaly bark. In cases where the initial injury involves very large patches of outer phloem but leavet the inner phloem and practically all the cambium intact, the resuls is approximately the same, excepting that occasionally small patches of bark die to the wood on account of the occurrence of coincident injured patches in the cambium and inner phloem. It often happens in instances of this kind that the cortex is affected but slightly and that it retains its normal appearance until the internal trouble has become far advanced; then it usually dies rather quickly and dries out. However, none of the types of injury so far described usually result in very serious trouble because at most only small areas of bark are killed to the wood.

When most of the cambium and much of the phloem are initially affected, the injurious results are usually much more evident; but even in such cases the bark may survive if the weather is favorable and if the area affected is not extensive as compared with the total area of the bark of that portion of the stem. In case the injured patches in the cambium and inner phloem are relatively large or fairly close together, or if they form nearly continuous sheaths of affected tissues, the regenerations from the living portions of the bark are hampered by defective water conduction outward from the wood; and unless the weather conditions are most favorable, so that the cells proliferating from the bark are able to re-establish living connections by fusing with the wood cylinder, these proliferating cells die, and the result is the death of the entire bark. Although such patches of dead bark are produced in various ways and result from injuries of varying degree, we term the wounds *crown-rot* if they occur on the bases of trunks or on roots near the ground, and *canker* if they occur on stems and branches above ground.

This histological investigation permits some inferences to be drawn that support most of the important contentions advanced in my former papers; as regards other contentions, however, the evidence is not so convincing. My preparations, as will appear later, indicate that both excessive tensions and certain degrees of immaturity of bark tissues have a causal relation to the development of the initial injuries that give rise to this bark disease. They also substantiate the results obtained by the cultural tests; no fungi are usually in evidence until the middle of May or even later.

The Development of the Disease

The first visible stage of crown-rot, as well as that of some other bark diseases, consists in a discoloration and collapse, or even in a rupture, of groups of tissues mainly of the inner bark. This stage is usually found only in late winter and spring, and is generally not evident to the ordinary observer unless the outer bark is conspicuously cleft. From late spring to mid-summer, however, most of the severe cases attract attention by the oozing of "sap" or gum and by the eventual discoloration of the outer bark. Such affected bark is most commonly found on the trunk near the ground, in crotches, and at the bases of small young branches arising from the large limbs of heavily pruned trees. When at or near the ground, dead bark rots quickly; above ground it usually dries and eventually scales off from the wood.

The initially affected tissues are variously distributed in streaks and patches, which in cross section usually appear in more or less nearly concentric circles about the wood cylinder. In cases of slight injury, the medullary rays of the inner phloem, groups of parenchyma around the sclerenchyma strands or patches of cortical parenchyma are affected. In more severe cases, much of the phloem and all the cambium may be involved or the phloem and cambium injuries may be accompanied by injuries in the cortex. The severity and course of the disease following such initial injuries depend upon the size and number of the affected patches, and upon the location of the most severely affected portions of bark.

The Initial Injuries.—Some of the common types of initial injuries that subsequently give rise to bark diseases are shown on Plate XXI, The group of figures shown on this plate does not, however, include one of the kinds most frequently noticed: these somewhat concentrically arranged injuries often occur with conspicuous radial clefts, as discussed in my former papers, and as indicated in Figs. 43 and 48, Plate VI. In many instances, however, the concentric injuries are not accompanied by radial ruptures, and sometimes radial clefts occur when other types of injuries are so slight as not to hinder subsequent normal bark growth. The sections shown on Plate XXI are all made from apple and pear material collected before growth started in spring (April 17, 1912, at Madison, Wisconsin).

Fig. 1 shows a condition that is frequently found in injured bark. Dead tissues are usually evidenced in these photographs by the occurrence of especially dark streaks or patches, by collapsed cells or by both. Sometimes ruptures are much more prominent than discolorations, as shown in Fig. 3, which shows a very common type of rupture or separation in the inner phloem. In Fig. 1 may be seen a conspicuous combination of the collapse of discolored tissues with ruptures in the inner phloem and cambium. At the left of the section shown in this figure the initial injury is confined chiefly to the cambium; on the right the principal injury occurs in the phloem, only scattered cells in the cambium being affected. A few groups of injured cells may also be seen about the sclerenchyma strands as well as farther out in the cortex. Fig. 2 shows a case from pear tissues in which the initial injury is most pronounced in the inner cortex and outer phloem, with only small groups of affected cells in the outer cortex and inner phloem. Fig. 3 is from apple. It shows a marked injury of medullary rays in the phloem, and a rupture of the phloem.

The other figures on Plate XXI show, on a larger scale, small areas in typically injured bark. In Fig. 4 occurs a mixture of streaks and patches of dead and living tissues present in the outer wood and the inner bark of apple. In the center of this figure a large ray and much of the surrounding tissue is dead and collapsed (appearing black). A portion of the continuation of this ray in the wood is also dead, although its cambial portion is still alive. In the next ray to the right, the outer portion is dead and that in the inner phloem and cambium is alive; but its extension into the wood cylinder is dead and discolored. The phloem and cambium tissues lying between these rays are mostly dead, but scattered living cells occur singly or in small groups. The phloem tissue between the two rays toward the right of the figure are in much better condition, containing only a few scattered groups of dead cells. It is worth noting that toward the right of the figure the main line or *sheath* of injury runs through the outer phloem, while toward the left it affects chiefly the cambium and inner phloem. However, even the most severely injured sheath has living cells interspersed among the dead and collapsed ones. In Fig. 5 such is not the case; here the sheath of injured tissue involves the cambium and inner phloem on the left; at about the middle of the figure it bends suddenly and proceeds through the phloem, leaving the cambium alive except for occasional groups of injured cells. In this instance the sheath of most severe injury contains few living cells, thus practically eliminating all living connection between the wood, or its living fringe of cambium, and the bark proper outside the injured sheath. Fig. 6 shows a comparable condition excepting that more dead cells are scattered among the living tissues. In this instance the broad ray near the center is dead in both bark and wood, and on the left the entire cambium and the inner phloem, with the exception of a few cells, are collapsed. The outer cortex shows many dead cells. Fig. 7 gives a better idea than the others of the injury occurring in the inner bark: here the cambium and inner phloem as well as the rays and much other phloem tissue are dead and more or less collapsed. In the cambial region near the left, however, is a group of two or three living cells with irregular outlines. These cells, as well as those of certain groups in Figs. 4 and 5, are apparently enlarged, although the apple trees from which this material was cut seemed perfectly dormant at the time. It appears likely that this represents the beginnings of spring growth and regeneration, brought on early as a response to the wound stimulus. In Fig. 8 occur two lines or sheaths of severe injury, one of which involves the cambium and the other the outer phloem. Neither of these zones is made up wholly of dead cells, so that the sheath of living phloem between them is not entirely without living connection with the wood and the outer bark. Fig. 9 shows a condition much like that in Fig. 7, excepting that larger masses of inner phloem are alive, although not evidently affording living links between the bark and the wood. In both Figs. 7 and 9 the sheath of dead tissue seems complete, thus isolating the bark from the wood cylinder at these places.

The initial injuries presented in Plate XXI are not shown because they represent the most severe cases, but because the location of the injuries is typical and yet they are not severe enough to prevent proper handling of the sections. As noted above, material for sectioning had to be taken from portions of trees where the areas of individual injuries or dead patches were relatively small in order to prevent the shattering of the blocks before they were imbedded. When small blocks were cut from the very edge of one of the more extensive injured areas, they frequently remained intact through the imbedding and sectioning processes; if, however, the entire block was within such an area, its different portions usually fell apart, separating along the planes of severest injury. This falling apart of the blocks was less troublesome in the material collected May 1 than in that collected April 17. The blocks cut on May 29 from the more severely affected and larger areas were extremely fragile, while those from regions of less injury were more stable than specimens of the same degree of injury collected April 17.

Some Changes Due to Growth and Regeneration.—The figures of Plate XXII are made from photographs taken of sections of apple collected May 1, 1912. These show some interesting phenomena of growth and regeneration, and among other things suggest how and why it is that so few bark injuries give rise to dead patches of bark.

In Figs. 10, 11 and 12 are shown cases in which the initial injury involved all or nearly all of the cambium and a portion of the inner phloem, with dead streaks of less extent scattered in other parts of the phloem. In all three of these cases subsequent regeneration growth from the living portions of the phloem resulted in establishing a more or less definite living connection through the zone initially involved. As a result of this growth, the material in the scattered dead streaks in other portions of the phloem has become compressed into ragged plates with their edges directed toward the wood. In other parts of the bark dead groups of cells are similarly compressed by the more or less bladdery growths from the surrounding tissues. In Figs. 10 and 11, comparatively few of the proliferating bark-cells appear to have established a close contact with the old wood; but in Fig. 12, representing a section in which the dead zone had not been so wide and still contained a few living cells, the new growth is much more firmly attached to the old wood. Fig. 13 again is more like Figs. 10 and 11, although its dead or injured zones are less extensive; in the section here shown, too, some living cells capable of further growth appear, which are attached to the old wood and have grown outward to meet the growth from the bark. In the tissues shown in Figs. 10, 11 and 12, the initial injuries had been most severe in the cambial and inner phloem regions and are of the type shown at the left in Fig. 1, Plate XXI; while Fig. 13 shows a regenerated condition of an initial injury more like that indicated in Figs. 7 and 9, Plate XXI, where some living bark cells or cell-groups remain attached to the wood. In Fig. 14 is shown an effort toward recovery that is rather remarkable and far advanced for May 1. This represents a reaction to wounds of the type shown near the right in Figs. 1, 3, 4, 5 and 6, Plate XXI. In addition to the general compression of the dead tissues by the growth of the living cells around them, many proliferating cells have pushed in among the portions of the dead sheath, thereby facilitating the re-establishment of living connections between the outer bark and the living inner phloem that remains attached to the wood-cylinder. Considerable injury also occurred in the cambium, although small portions of the latter appear to have survived. A new cambium, however, is seen to be forming outside among the irregular cells arising from the wound growth. The figure does not show it as clearly as the microscope; it is beginning to take form in the line *cc*. Fig. 15 represents a similar instance, except that the initial injury was more extensive and that larger groups of dead cells resulted. A new cambium is forming at *cc*, though it is incomplete and still has compressed fragments of dead tissue in its course. Fig. 16 seems to be a later stage of a case something like the left-hand portion of Fig. 8, where the cambium was only slightly injured and the outer phloem rather severely, though in more or less isolated streaks and patches. Some of the rays are dead, although a few are practically normal, like that near the right of Fig. 16. The new cambium is quite distinctly indicated by the dense band *cc*. In Fig. 17, comparable but severer initial injuries obtained. The re-established living connections between the growing phloem and the wood are few and scattered, and the injury in the outer phloem forms a nearly complete sheath, thus isolating the cortex considerably from the phloem. A scattering of living cells occurs, however, in this outer-phloem zone to afford water and nutrient transfer between the outer and inner barks sufficient to permit the outer bark to endure at least for a time.

The figures of Plate XXII likewise give only a few of the great variety of the injuries that were mild enough to permit more or less regenerative growth during the spring, although some of the figures plainly indicate that only a most favorable summer would enable the affected bark to survive.

Some Results Found at the End of May.—Plates XXIII and XXIV give an idea of the great variety of results following some of the initial injuries shown on Plate XXI. The low-power views collected on Plate XXIII make it evident that regeneration and growth of the living cells are not all that is required to sustain the affected bark and to keep it from dying in early summer. Figs. 18 to 22 inclusive show some of the milder forms, while Figs. 23 to 26 indicate various stages and degrees of injury resulting in the death of patches of bark.

Fig. 18 shows an advanced stage of an initial injury of the type shown in Figs. 7 and 9, and, in later condition, in Figs. 10 and 11. The new cambium sheath arose much after the manner shown in Fig. 17. The compressed fragments of dead tissues, present at the time spring growth started, are noticeable in the new wood (*nw*) as well as in the old phloem (*op*). The initial injury was so severe that the old wood and bark-rays were not continued by the new growth; new rays are just becoming differentiated on both sides of the new cambium (*nc*). In this case the outer bark seems to have established sufficient living connection with the regenerating inner bark to continue its normal functioning, but the connection between the old (*ow*) and the new wood (*nw*) seems to be insufficient in places, for the new cambium (*nc*) has also developed in the inner phloem. In this case the initial injuries in the outer phloem and cambium were so extensive as greatly to delay the development of the new cambium (*nc*), as seen near the middle of the figure. No definite new wood cells have yet been formed at this point, although on both the right and left sides a considerable layer of new wood has resulted and the new cambium appears practically normal. Fig. 20 shows some interesting irregularities in the distribution and configuration of the initial injuries. They had evidently been of the type shown in Figs. 1, 5 and 9, and the new cambium apparently started to form in the inner phloem just outside the dead cambium as shown by the light line near the wood, at the lower left. But because of marked irregularities in the distribution of the sheath of severest injury, the first new cambial initial was supplanted by one farther removed from the wood and overtopping the irregularities, as shown at *nc*. Toward the left of this figure the injury had been so severe as wholly to inhibit the development of a new cambium, although the cell proliferation had occurred that ordinarily precedes the production of a new cambium. Some of the small groups of living cells in that region had become light brown in color and were evidently dying. Toward the left end of the new cambium only scattered groups of new wood cells had developed. The portion of old inner phloem, separated from the rest by the mantle of injured tissue (near the center), had grown somewhat, but no definite cambium had been evolved. In the section shown in Fig. 21 similar isolated strips had developed into wood cells, even though the new cambium, as in Fig. 20, developed farther out in the phloem and produced a rather broad layer of new wood outside the zone of greatest injury. This type of figure seems to have been developed as the result of initial injuries like those shown in Figs. 5, 7 and 9. Much dead tissue was compressed into irregular masses in the outer phloem and cortex. The new cambium (*nc*) has a brownish tinge and seems to be much collapsed. It should be noted in this case that the phloem left attached to the old wood was transformed into wood without leaving a cambium. Fig. 22 is interesting chiefly on account of the fact that injuries in the outer cortex resulted in the development of a new phellogen layer or cork cambium within (*ph*). Fig. 2, Plate XXI, shows an injury occurring mainly in the inner cortex, that is often similarly cut off by a phellogen developing in the outer phloem. Fig. 23 is somewhat comparable to the left portion of Fig. 20, in that no new cambium has developed, although considerable regeneration growth has occurred. The cortex is practically uninjured and therefore appears normal from the outside, but both the outer and inner phloem are severely affected and the cambium is entirely dead, except in isolated streaks like that shown near the left. But even in this severely injured phloem occur groups of living cells, though they are more or less completely isolated by dead tissues. Many enormous bladdery outgrowths from the living cells are forced into the dead masses. In some places the living and in others the dead tissues predominate in the phloem. It may be noticed that on the left the bark is thicker than it is on the right. This results from the presence of larger groups of living cells in this portion of the phloem; indeed, it appears that groups of wood cells (appearing in the photograph as rather dim whitish patches) have in some way arisen in this region. The process had advanced further in the specimen shown in Fig. 24. Here some of the bark had died to the wood, and, because of the presence of much dead tissue in the inner phloem and the old cambium, the callus is a rather sickly affair. It includes the repaired phloem considerably speckled with masses of dead tissue, and the badly injured cortex. In the lower right corner occurs a strip (white) where the phloem is being transformed into wood cells, yet no definite cambium is in evidence. Although not shown in this figure, the slide from which this photograph was made shows abundant hyaline fungus mycelium in the dead bark, even in the dead cambium and the old inner phloem between the callus and the old wood. Fig. 25 shows a similar case in which the most severely affected area was very narrow, thus permitting its use in this study without its falling apart. In this instance the callus is much better developed, having a definite cambium and a layer of new wood. The old inner phloem and the old cambium were also dead for some distance back of the nose of the callus. Some fungus mycelium was present in the dead bark. Fig. 26 is made from the margin of a larger area that had sunken in, like that shown in Fig. 41 of Plate XXV. The marginal callus was much like that of Fig. 25, and the presence of fungus is indicated by the pycnidia (of *Sphaeropsis?*) showing under the periderm toward the left.

On Plate XXIV are brought together some higher-power views giving greater detail, though in some instances cell outlines are necessarily more hazy. Figs. 27 and 28 show clearly the remains of the dead cambium and inner phloem; they also prove that even as late as the end of May substitution growth is in progress along the inner side of the repaired bark and has established better connection with the old wood. This latter fact is indicated by the presence of excessively large round cells, that appear to be filling the gaps left by the shrunken dead tissues. Fig. 30 shows the development of a new cambium (*nc*) between the dead inner phloem and sheets of dead tissue in the outer phloem. In Fig. 31, groups of dead cells appear in the former position of the inner phloem, around and among which some of the new wood (*nw*) cells are becoming discolored. This intermingling of living and dead groups of cells is most common in the phloem. The position of the new cambium (*nc*) is occupied chiefly by structureless masses having a yellowish brown color. The cortex and outer phloem had only scattered groups of dead cells. No fungous hyphae could be detected in this specimen. Fig. 32 shows tissues which had sustained more severe initial injuries but which, because of the small area of the most severely affected part, endured quite well, while in the case of milder injury but of larger area shown in Fig. 31 the tissues seemed to be dying. In the tissues shown in Fig. 32, the outer phloem also is largely killed but the cortex is in fair condition. A new cambium (*nc*) is forming near the old wood. At this place no living connection appears between the bark and the wood, but because of the smallness of the region so severely affected, the necessary water and nutrients seem to reach it from the sides of the injured patch where such connections do occur. Fig. 33 shows a case in which much of the inner phloem had been killed and in which very active filling-growth is occurring. A faint indication of the formation of a new cambium can be seen here and there; a spot of this kind appears near the center of the figure. The photograph from which Fig. 34 was made shows a very large group of dead phloem tissue which has been only partly permeated by proliferating cells arising from living adjoining cells. In some cases the initially killed strips of tissue form an anastomosing network lengthwise through the bark; in extreme instances only anastomosing streaks of inner bark may be alive while the greater mass of the bark is dead. A cross section of such a living streak in great masses of dead tissue may be seen in Fig. 35. In this instance large groups of dead cells also occurred in the inner cortex, although when the specimen was cut (May 29) its external appearance was practically normal.

From another type of initial injury enlargements as well as wood-exposures occur on trunks of trees. Such cases of excessive enlargements on trunks may sometimes develop from initial injuries of the type shown in Fig. 3, Plate XXI, in which a definite separation or a concentric cleft has resulted, and where at the same time the repair growth and connection with the wood are such as to prevent the dying of the loosened outer bark. When radial clefts run through such areas and the bark is otherwise sufficiently intact to withstand the drying action of air, the repair growths may turn the edges of the loose bark outward, while if no radial cleft occurs the enlargement may look like that on a maple trunk shown in Fig. 40, Plate XXV. The resulting repair growths are not always uniform even when they follow the kind of initial injury that separates the bark from the wood. In some cases the loosened bark has but few injuries (Fig. 3, Plate XXI); in other cases, or perhaps even in other portions of the same affected area, the outer phloem as well as the cortex may have so many groups of dead cells scattered among the living parts that the entire bark dies. That has been the case in the lower portion of the stem part shown in Fig. 40. A section through the upper part of this maple trunk reveals a condition like that shown in Fig. 37. Here the discolored line *oc* represents the position of the cambium when the injury occurred. The initial injury not only resulted in a line of separation in the inner phloem like that shown in Fig. 3, but involved cell-groups in the cambium proper as well as in the middle and outer bark. As in the case shown in Fig. 3, however, the inner phloem had sufficient living connection with the wood to permit the development of a cambium that persisted, excepting in the bare region shown toward the lower end of Fig. 40, where it died along with the loosened bark outside. A cambium also developed in the inner part of the loosened bark shown in Fig. 3, running through the outer phloem. After the production of a sheath of new cells in this new growing zone, the middle ones became wood and those along *both outer* sides continued as cambial zones. In that way *one* growing zone was converted into *two*, which separated more and more as the older cells toward the middle were converted into wood. The sheath of new wood (*nw*) just within the old outer bark (*ob*, Fig. 37) arose in that manner, and has a cambium on each side. The low-power views shown in Fig. 36 (*a–e*) are photographs of sections cut from blocks obtained from the specimen shown in Fig. 37. Fig. 36*a* shows the old outer bark (*ob*) of Fig. 37, with only a small portion of the new cambium (*nc*) included. The new bark shows compressed inclusions of dead tissues resulting from the initial injury. Figs. 36*b* and 36*d* are so tightly pressed against each other in Fig. 37 that the two barks seem to be one. Fig. 36*e* is taken from the line *oc* in Fig. 37 and shows more clearly the similarity of this line to the figures obtained from apple, such, for example, as Fig. 18. Fig. 39 is a more highly magnified view of a portion of Fig. 36*e*. It shows the presence of dead groups of old cambium cells adhering to the old wood (*ow*).

It is evident that in most places along the line *oc* of Fig. 37 the cambium, giving rise to the new wood (*nw*) outside it, also arose much like that shown in Figs. 14, 15, and 17, from cell divisions occurring in the adhering layer of phloem (Fig. 3). Trécul[4] seems to have obtained very similar results by pulling loose and then tying back a piece of bark.

Fig. 39 shows another fact of special interest, for it makes it apparent that much of the new wood formed about the original line of injury subsequently dies and becomes discolored. Even in portions of the line *cc* of Fig. 37, where the new wood is practically continuous with the old and where the rays are not even interrupted, there has been more or less of this discoloration and dying of the new wood, as shown in Fig. 42. Here the initial injury seems to have involved chiefly the outer end of the wood rays and scattered, but small, groups of cambium cells. Nevertheless a narrow, ragged-edged zone of new wood died and became discolored, as Fig. 42 shows.

The Development of Crown-Rot from the Initial Injuries.—As suggested above, bark-injuries may or may not be accompanied by evident radial clefts, and when they are not evident externally they are yet often present, as may be gathered from Fig. 36*a*. The old bark (*ob*) is shown to be run through by numerous small, radial rifts that represent incomplete ruptures which were afterwards filled by proliferations from adjoining cells. Fig. 38 shows a case of this kind, also in maple, where apparently the bark was only separated from the wood, and yet where so many of these tiny radial rifts occurred that the bark involved is evidently dying. In this case the whole of the cambium as well as much of the inner phloem died as a result of the initial injury. The rest of the bark was still alive when cut on May 28. Although no definite new cambium had yet developed in this loosened portion of bark, the spring growth of wood is seen to be considerable on both sides of the wound. Callus-roll formation has made an evident beginning around the injury, even though the bark involved is not dead.

On the other hand, Figs. 43 and 45, Plate XXVI, show apple trees in which internal bark-injuries, resulting in a separation of the bark from the wood, were accompanied by evident radial clefts. The former is shown before and the latter after the loose bark was removed. Fig. 44 shows the same tree as that in Fig. 43 with the loose bark removed, making it apparent that the tree was half girdled, though only a fairly narrow band (not exceeding 17 cm. in vertical width) was involved. The loosened bark shown as removed in Fig. 45 had several short radial clefts, though otherwise the bark appeared normal while it was still on the tree. When removed, the inner surface of the bark and the wood thus exposed had a rusty brownish color. On cutting with a knife it was found to contain numerous closely scattered, small dead spots, which in some places had coalesced to form ragged, dead patches as much as one or two centimeters in diameter. These patches often involved all but the outermost layers of the cortex and sometimes showed through the periderm or scaly outer bark in the form of dead spots from one to five millimeters in diameter. In places, however, this loose bark was found to have developed a new cambium in addition to bladdery outgrowths from the inner phloem, thus tending to re-establish connection with the wood cylinder. Similar, though scattered, outgrowths had also developed from outside the wood cylinder, but the actual connection established was evidently slight; for on June 24, when it was removed, considerable areas of these proliferations had died and turned a rusty brown. In fact, disorganization seemed to have set in over a large part of the inner surface of this bark.

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[4] Trécul, A., Production du bois par l'écorce des arbres dicotylédonés, Ann. Sci. Nat. Bot. III **19**: 257. 1855.

When, on May 7, the loosened bark was removed from the tree shown in Figs. 43 and 44, only a slight discoloration was noticed on the contact surfaces. The loosened bark appeared perfectly normal on the outside, with the exception of the presence of a wide radial cleft. Numerous scattered groups of dead tissue were found in the older phloem and inner cortex. Proliferation growth had been abundant, and in a few places it appeared that cambium was in process of formation.

In cases of injury in which the affected bark does not die and where but *one* substitute cambium develops, only the discolored line in the wood afterward remains as a permanent record. This line is marked *oc* in Figs. 18 and 19, Plate XXIII, and 37, Plate XXV. Fig. 49, Plate XXVII, is an especially clear illustration. It represents a cross-section of the base of a large apple-tree trunk from an orchard in which the initial injuries, leading to the development of crown-rot, had occurred on many trees the same number of years back, as is indicated by the radial-growth zones outside the conspicuous line of discoloration in the wood. It should be noted also that at least one radial cleft occurred in the bark of the tree represented in Fig. 49: the fusion is still incomplete. However, in the tree, a section of which is shown in Fig. 50, a portion of the affected bark died and the entire wood cylinder, up to the line represented by *oc* in Fig. 18 (the outer boundary of the wood at the time of injury), has completely decayed. In a few points, just as in Figs. 3, 39 and 42, some of the wood produced afterward also died and decayed. This shows that decay depends as much upon the death of the wood as upon the presence of wood-rotting fungi. Bark and wood having groups or streaks of dead tissue naturally contain relatively large quantities of air, and sapwood dying from such bark injuries is full of both air and stored food. The high air content of such wood led Münch[5] to conclude that the presence of the excessive air is the factor that permits wood-rotting fungi to vegetate actively in such tree trunks. Based on these conclusions of Münch, Appel[6] has been led far afield in his discussion of the factors governing the activity of wood-rotting fungi. The fact that such wood is killed while it is young and full of stored food makes it evident that it differs materially from ordinary heartwood that has become depleted of most of its stored food (including the layers of hemicellulose usually present on the inside of its cell-walls) before it became lifeless. It seems more likely that wood-rotting fungi thrive uncommonly well in such wood because it contains large quantities of stored food and masses of more or less disorganized and therefore non-resistant protoplasm, rather than because of the great abundance of air present.

The small apple tree shown in Fig. 46 and the large ones of Figs. 47 and 51 are examples in which the most severely affected bark died. In those shown in Figs. 26 and 51 a complete girdle is involved, while in that of Fig. 47 only about three fourths of the bark succumbed.

Comparison of Effects on Large and Small Trees.—The initial injuries, from which crown-rot and some other bark diseases arise, are the same on large and small trees; the differences usually noticed afterward result from subsequent changes owing to differences in the thickness of the bark and in the diameter of the stems or branches involved. It is also shown that if the area of the bark most severely affected is large compared with the circumference of the stem involved, the result is more serious than when the injured patch is comparatively small. This holds for both large and small, as well as for young and old stems. If the section from which Fig. 26 is made were photographed whole and magnified, a picture much like Fig. 50 would result, although the wood cylinder within the injured zone was not decayed or even entirely dead when cut on May 29. The new growth of wood shown in Fig. 26 as well as that in Fig. 25 consisted of only a very thin layer, while the wood shown in Figs. 49 and 50 represented several years' growth. Fundamentally, however, these sections are not only comparable but very similar.

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[5] Münch, E., Untersuchungen über Immunität und Krankheitsempfänglichkeit der Holzpflanzen, Naturw. Zeit. Forst. Landw. 7: 54; 87; 129. 1909.

[6] Appel, O., The Relations between Scientific Botany and Phytopathology, Ann. Mo. Bot. Gard. 2: 275. 1915.

The Cause of the Initial Injuries

The work so far has clearly shown that the initial injuries in the bark of trees that result in crown-rot arise during the dormant season, but their cause has not been definitely established. The years of observation and a few experiments together with the histological study here reported, indicate the most probable factors, and thus pave the way for an experimental study of the problem. In general terms it may be said that these initial injuries are due to a lack of adjustment between radial or bark-growth and the environment.

Some Facts about Bark-Growth as Related to the Development of These Injuries.—In the study of forest trees it has been shown that growth and development proceed in a wave-like manner. The various functions, the size of cells, and the amount of annual growth increase to maxima in certain stages of a tree's life, and decrease to minima again at other stages. These periods or cycles are repeated at intervals more or less characteristic of a species. Kapteyn[7] calls attention to growth cycles that may be traced in the wood and extend over periods of 12.4 years, apparently independent of the species. Bailey and Shepard[8] found that the length of coniferous tracheids varies in more or less definite cycles usually ranging from 35 to 80 years, and apparently differing in different species.

It is a well-known fact, for instance, that at a certain age of a woody plant the development of primary bark is supplanted by the formation of secondary bark, apparently because of the inability of the primary tissues to continue their adjustment to the increase of the stele. This transition stage in trees usually occurs at an age characteristic for the species. Afterward the bark continues to increase in thickness, and to be distended, growing somewhat radially for a certain number of years; then a new phellogen is again formed inside that portion of the bark that is no longer able to undergo sufficiently rapid tangential growth, and a fresh portion of lifeless bark results which is more or less continuous around the trunk—the results in this respect also varying with the species.

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[7] Kapteyn, J. C., Tree-Growth and Meteorological Factors, Réc. Trav. Bot. Néerland. 11: 70. 1914.

[8] Bailey, I. W., and Shepard, H. B., Sanio's Laws for the Variation in Size of Coniferous Tracheids, Bot. Gaz. 60: 66. 1915.

It appears that there is a close relation between the growth cycles mentioned above and the periodicity that may usually be noticed in regard to bark cycles. The growth and cell-size minima seem usually to fall in the season just preceding a new period of bark-roughening, while the maxima are usually reached during the second third of the time elapsing between the occurrence of two minima. The environmental variations, and, in case of cultivated trees, the culture and tilth given, have a marked influence upon the prominence of this periodicity.

In a smooth-barked stem that portion of the bark outside the oldest circle of sclerenchyma (the cortex) often undergoes much, although limited, growth. Sections of apple and pear stems from material fixed at different times of the year indicate that during seasons of much or of late radial growth cortical growth sometimes continues very late and is not completed by the time the period of dormancy arrives. The increase in diameter of wood necessitates and *is followed by* an increase in the area of the bark. When an adverse change in the weather conditions interferes before this cortical growth is completed, the dormant period must be passed with the outer bark in this unfinished condition. In such a case the bark is often under considerably higher transverse tension than it is in cases in which its cortical growth has been finished. In instances also in which bark growth has been very slight during some years, the cell walls of the cortical tissues and those in the outer phloem are thickened to such an extent that a rather rapid resumption of radial growth is not immediately followed by cortical growth, and therefore high bark tension ensues. If such hardened outer bark is eventually forced into growth late in the season, some of the cells necessarily pass through the dormant season in immature condition, and thus are likely to become injured. Nördlinger[9] found by peeling tests that cambial activity precedes cortical growth and may continue after cortical growth ceases. In some cases, however, cortical growth continued later than cambial activity.

R. Hartig[10] describes several cases in which the bark of a very high percentage of forest trees was burst and injured at certain places two years after those forests had been thoroughly thinned. After thinning, the trees grew as much in one year as they had grown before in many years. His conclusion, that the bark burst in early summer owing to the rapid radial growth of the wood, can fortunately be more carefully examined because he gives a photographic record of cross-sections. These figures prove beyond question that the bark was split and separated from the wood during the *dormant season preceding* the growing season in which he assumes the splitting to have occurred. From Fig. 52, Plate XXVII, which is a reproduction of one of Hartig's figures, it is apparent that the bark injury occurred *between* the growing seasons and not while growth was going on because the lines of injury and separation coincide with the line separating the wood of two growing seasons. Another case which Hartig gives in some detail, in which a high percentage of the trees in a thinned forest sustained bark injuries just above or at the ground line a few years after thinning, is also of decided interest. In this instance he concludes that the rank growth of herbaceous plants developing about the tree trunks after thinning prevented proper aeration, excluded light, and thereby injured the bark. But in this case as in the former, cross-sections show that the injury occurred during the dormant season, when aeration was probably good. The chief difference between these two cases lies in the fact that in the former instance the tension reached a high enough point to rupture the bark as well as to loosen it, while in the latter the tension was less. It seems possible that in one instance the bark was more resistant to radial rupture than in the other, though it is likely that some additional factors are involved in the occurrence of radial clefts.

[9] Nördlinger, H., Wann beginnt Bast, wann Lederschicht der Rinde sich zu lozen? Centralbl. Gesamt. Forstwes. Wien. 5: 128. 1879.

[10] Hartig, R., Zersprengen der Eichenrinde nach plötzlicher Zuwachssteigerung, Untersuch. Forstbot. Inst. München 1: 145. 1880.

——, Das Zersprengen der Hainbuchenrinde nach plötzlicher Zuwachssteigerung, Untersuch. Forstbot. Inst. München 3: 141. 1883.

Environmental Factors Having a Causal Relation to the Injuries.—My work in northern orchards showed that bark injuries which are caused by the combination of immaturity of tissues and the occurrence of low temperature may give rise to crown-rot. In Florida the occurrence of a temperature only about two degrees or less below the freezing point of water, when certain bark tissues of citrus trees are immature, may result in similar injuries and give rise to equally destructive diseases. Severe droughts often cause similar injuries. When the bark of citrus trees is dormant, it will endure temperatures even below – 10° C. and severe droughts without serious injury.

It is still uncertain whether this bark injury is due chiefly to simple physical causes, such as contraction or to chemical and physiological changes induced in the protoplasm by low temperatures and drought, or to both sets of factors acting together. As a matter of fact, changes of both kinds take place in plants subjected to low temperature and untimely droughts, and we have fairly tangible evidence that both may be injurious.

The photographs submitted with this paper give ample evidence that high tensions, and even ruptures, accompany some at least of the more severe bark injuries. Trunk measurements previously published also show the occurrence of high tensions.[11] In some cases, however, no actual ruptures appear to result, and yet tissues become discolored before the commencement of the next vegetative period. Injurious low-temperature tensions of less degree than those required to rupture the bark are evidently of frequent occurrence, and these are apparently responsible for much of the bark injury afterwards resulting in disease. An extreme form of this effect is shown in Fig. 37*a*. Some of the milder tensions are also shown in Figs. 3, 4 and 5 of Brown's[12] recent paper. Sorauer[13] has given much attention to this type of injury and even applies his low-temperature tension hypothesis to cold injury of herbaceous plants.

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[11] Crown-Rot of Fruit Trees: Field Studies, N. Y. Agr. Exp. Sta. Tech. Bull. **23**: 36. 1912.

[13] Sorauer, P., Experimentelle Studien über die mechanischen Wirkungen des Frostes bei Obst- und Waldbaümen, Landw. Jahrb. **35**: 469. 1906.

——, Weswegen erkranken Schattenmorrellen besonders leicht durch Monilia? Zeit. Pflanzenkr. **22**: 285. 1912.

——, Einige Experimente zum Studium der Frostwirkungen auf die Obstbäume, Die Naturw. **1**: 1055; 1094. 1913.

[12] Brown, H. P., Growth Studies in Forest Trees. 2. Pinus Strobus, Bot. Gaz. **59**: 197. 1915.

——, Altes und Neues über die mechanischen Frostbeschädigungen, Zeit. Pflanzenkr. **24**: 65. 1914.

Some interesting advances have been made in recent years in the study of the chemical and physiological side of this question, but unfortunately the investigators interested in this phase of the subject have thus far given no attention to the more simple physical concomitants presented by Sorauer in the papers just referred to. It is in fact usually assumed that the earlier works had decided this matter. Nägeli,[14] for example, made some studies of this type and concluded that since walls of Spirogyra cells killed by low temperature are not ruptured, death must be due to changes induced in the protoplasm. Kunisch[15] maintained that low temperatures induce harmful irreversible changes in certain components of the protoplasm that result in the death of tissues; that in some plants such changes may even occur above the freezing point,[16] although in others a temperature much below freezing is necessary to cause injurious effects. Fischer,[17] after very fully discussing the literature and giving the results of his own extensive experimental study of the problem, concluded that the low-temperature death-point of plants usually does not vary more than two, though it may vary as much as ten, degrees. On the other hand, Winkler[18] found that the condition of the protoplasm at the time of the occurrence of the low temperature has much to do with the degree of resistance or injury.

Some have held that low-temperature injury results from ice-formation; others believe that it is the withdrawal of water during freezing that causes the injury.[19] Apelt and others[20] have brought

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——, Über Frostschorf an Apfel- und Birnenstämmen, Zeit. Pflanzenkr. **1**: 137. 1891.

[14] Nägeli, C., Ueber die Wirkung des Frostes auf die Pflanzenzellen, Sitzungsb. Akad. Wiss. München **1**: 264. 1861.

[15] Kunisch, E. H., Ueber die tötliche Einwirkung niederer Temperaturen auf die Pflanzen, Inaug. Dissert. Breslau. 1880.

[16] Molisch, H., Untersuchungen über das Erfrieren der Pflanzen. Jena. 1897.

——, Das Erfrieren von Pflanzen bei Temperaturen über dem Eispunkt, Sitzungsb. Akad. Wiss. Math. Naturw. (Wien) **105**: 82. 1896.

[17] Fischer, H. W., Gefrieren und Erfrieren, eine physico-chemische Studie, Beitr. Biol. Pflanz. **10**: 133. 1911.

[18] Winkler, A., Über den Einfluss der Aussenbedingungen auf die Kälteresistenz ausdauernder Gewächse, Jahrb. Wiss. Bot. **52**: 467. 1913.

[19] Müller-Thurgau, H., Ueber das Gefrieren und Erfrieren der Pflanzen, Landw. Jahrb. **9**: 133. 1880.

out some very pertinent reasons for their contention that low-temperature injury is not due to the withdrawal of water.

Even in much of the older literature on this subject, one finds interesting comments on the apparent changes that living substances and stored foods of plants undergo on the gradual approach of cold weather. It was noted, too, that plants thus modified are more resistant to low temperature. The more modern work on this phase of the subject, by Gorke[21] and Lidforss,[22] as well as the work by Bartelzsko,[23] formed a basis for the most interesting researches of Maximow.[24] The latter author showed that it is not the molecular concentration of the cell sap that is most significant, but that the physical nature of the solutes determines the degree of resistance afforded a plant. It was found that the introduction into plant

Greeley, A. W., On the Analogy between the Effects of Loss of Water and Lowering of Temperature, Amer. Journ. Physiol. **6**: 122. 1902.

Matruchot, L., et Molliard, M., Modifications produites par le gel dans la structure des cellules végétales, Rev. Gen. Bot. **14**: 463; 522. 1902.

Bruijning, F. E., Zur Kenntniss der Ursache des Frostschadens, Forsch. Gebiete Agr. Phys. 19: 485. 1896.

Chandler, W. H., The Killing of Plant Tissue by Low Temperature, Mo. Agr. Exp. Sta. Research Bull. **8**: 143. 1913.

[20] Apelt, A., Neue Untersuchungen über den Kältetod der Kartoffel, Beitr. Biol. Pflanz. **9**: 215. 1909.

Rein, R., Untersuchungen über den Kältetod der Pflanzen, Inaug. Dissert. Halle. 1908.

Voigtländer, Hans, Unterkühlung und Kältetod der Pflanzen, Beitr. Biol. Pflanz. **9**: 359. 1909.

Mez, Carl, Neue Untersuchungen über das Erfrieren eisbeständiger, Pflanzen, Flora **94**: 8. 1905.

[21] Gorke, H., Über chemische Vorgänge beim Erfrieren der Pflanzen, Landw. Vers. Stat. **65**: 149. 1907.

[22] Lidforss, B., Die wintergrüne Flora, eine biologische Untersuchung, Lunds Universitets Årsskrift, N. F. 2^2, nr. 13. 1907.

[23] Bartelzsko, H., Untersuchungen über das Erfrieren von Schimmelpilzen, Jahrb. Wiss. Bot. **47**: 57. 1910.

[24] Maximow, N. A., Chemische Schutzmittel der Pflanzen gegen Erfrieren, I. Bericht. Deutsch. Bot. Ges. **30**: 52. 1912.

——, Chemische Schutzmittel der Pflanzen gegen Erfrieren, II. Die Schutz. wirkung von Salzlösungen, Bericht. Deutsch. Bot. Ges. **30**: 293. 1912.

——, Chemische Schutzmittel der Pflanzen gegen Erfrieren, III. Über die Natur der Schutzwirkung, Bericht. Deutsch. Bot. Ges. **30**: 504. 1912.

——, Experimentelle und kritische Untersuchungen über das Gefrieren und Erfrieren der Pflanzen, Jahrb. Wiss. Bot. **53**: 325. 1914.

tissues of substances having relatively high cryohydric points gave very little added resistance even though their molecular concentrations were high, while the introduction of substances with very low cryohydric points afforded much added resistance, even at fairly low concentrations. He concluded that since the low-temperature death-point can be lowered by the introduction of substances of low cryohydric points, protoplasm can have no specific death-point, but that the death-point depends upon the temperature at which water and other substances are crystallized out. Some interesting experiments by Gassner and Grimme[25] also show that Maximow's results have a wide application.

The part played by enzymes in plants injured by low temperatures is still rather uncertain, though they are probably involved in the many protoplasmic changes that result. It seems very likely, too, that some of the harmful changes that are caused by low temperatures are due to the perverted action of enzymes no longer properly controlled by substances that have been modified by the cold. Krasnosselsky[26] found that an oxidizing enzyme evinced more activity in sap expressed from a frozen plant than in that obtained from living tissues. The browning of sap expressed from tissues injured by cold is suggestive of the brown-spotting of herbaceous plants obtained by Molisch in experiments cited above, in which low temperatures above the freezing point were used. Möbius[27] obtained very similar results. At any rate, it has been well established that the best known enzymes present in plants are not destroyed by ordinary low temperatures, for Palladin[28] and his students use low temperatures to kill tissues before extracting enzymes. Kovchoff[29] maintains that protein-splitting enzymes are very active in cold-injured plant tissues, though his experiments seem to admit the assumption that perhaps the proteins were split as a direct result of the low temperature and that the changes he records were mainly due to the subsequent increase in the activity of the oxidizing enzyme present in the injured tissues. From his experiments with low temperatures, Schaffnit[30] holds that temperatures a little above the freezing point induce chemical changes in the protoplasm that convert labile into more stable compounds.

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[25] Gassner, G., und Grimme, C., Beiträge zur Frage der Frosthärte der Getreidepflanzen, Bericht. Deutsch. Bot. Ges. **31**: 507. 1913.

[26] Krasnosselsky, T., Bildung der Atmungsenzyme in verletzten Pflanzen, Bericht. Deutsch. Bot. Ges. **23**: 142. 1905.

[27] Möbius, M., Die Erkältung der Pflanzen, Bericht. Deutsch. Bot. Ges. **25**: 67. 1907.

[28] Palladin, W., Über den verschiedenen Ursprung der während der Atmung der Pflanzen ausgeschiedene Kohlensäure, Bericht. Deutsch. Bot. Ges. **23**: 240. 1905.

[29] Kovchoff, J., Enzymatische Eiweisszersetzung in erfrorenen Pflanzen, Bericht. Deutsch. Bot. Ges. **25**: 473. 1907.

The discoloration noted in the new cambium of Fig. 31, Plate XXIV, and in the new wood shown in Figs. 39 and 42, Plate XXV, is probably due to the diffusion into the new tissues of some injurious by-products from cells affected by the cold. This aftermath of low temperature effects seems to account for the fact that bark-injured trees may be sick for some time before they die or recover, and that some may remain dwarfed for years.[31] Münch[32] found that the discoloration so commonly present in the heart wood of some trees is not due to substances secreted by living cells, but to oxidation products arising in dead cells. The presence of fungi increases the extent of the browning. Goethe[33] has also made notes on this diffusion into living tissues of an injurious substance from tissues injured by low temperatures. He found that even in cases of severe bark-injury on the lower portions of tree-trunks, the affected trees survived if this discolorizing substance did not diffuse throughout the sapwood, while if its diffusion was rapid and extensive the tree usually died in a fairly short time. The same facts were found to hold regarding branches at the crotches of which bark-injury had occurred. Sorauer[34] thinks it likely that the injurious substance which diffuses from dead protoplasm into surrounding cells is an enzyme which arose from the disintegrating protoplasm. Active growth is said to check this diffusion or to make it harmless. Bailey[35] found, also, that oxidizing enzymes are largely responsible for discolorations developing in new green lumber during warm, moist weather.

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[30] Schaffnit, E., Studien über den Einfluss niederer Temperaturen auf die pflanzliche Zelle, Mitth. Kaiser. Wilh. Inst. Landw. Bromberg 3: 93. 1910.

[31] Gutzeit, E , Dauernde Wachstumshemmung bei Kulturpflanzen nach vorübergehender Kälteeinwirkung, Arbeit. Biol. Anstalt Lands. Forstwirts. 5: 449. 1907.

[32] Münch, E., Über krankhafte Kernbildung, Naturw. Zeit. Forst-Landw. 8: 533; 553. 1910.

[33] Goethe, R., Die Frostschäden der Obstbäume und ihre Verhütung. Berlin. 1883.

[34] Sorauer, P., Was bringen wir mit den Samenrüben und Samenknäueln der Zuckerrüben in den Boden? Zeit. Pflanzenkr. 24: 449. 1915.

[35] Bailey, I. W., Oxidizing Enzymes and Their Relation to Sap Stain in Lumber, Bot. Gaz. 50: 142. 1910.

It seems possible, too, that certain degrees of severity in the environment disturb the equilibrium between the enzymes in cells that are in a susceptible condition, and thus eventually lead to disintegration which may culminate in the death of the tissues. Such an assumption might lead to the surmise that the disintegrations evident in the cambial region shown in Fig. 31 are due to an excess of a hydrolyzing enzyme or to the absence of factors that normally inhibit hydrolytic action at a certain stage of growth, and permit the usual maturing processes to go on to completion. Lepeschkin's[36] studies of the effects of high temperatures on protoplasm, as well as some of the results noted by Overton[37] when using heat to kill portions of Cyperus stems, are interesting in this connection because they suggest the possibility that opposite extremes of temperatures may, after all, have some parallel effects.

Although the researches that have been cited on the chemical and physiological phases of low-temperature injury are apparently of fundamental importance, they give only a very meager understanding of what seems to be a small portion of the process. As already mentioned, some of the simpler physical effects of a lowering of the temperature must also be brought into proper relation with the physiological changes induced. After these simpler matters have been disposed of and a fair understanding of the development of bark injury has been attained, the practical phases of the problem will still be unsolved. One who has given this subject much thought cannot avoid the striking fact that *in nature* these injuries ordinarily occur not so much on account of the *degree* of the low temperature reached, as because of the *condition of the bark* at the time of its occurrence.

SOME OTHER BARK DISEASES RESULTING FROM INTERNAL BARK INJURIES

In the course of my study of crown-rot some other bark diseases were also traced to their origin in bark injuries very similar to those often giving rise to crown-rot. The so-called "cankers," "sun-scorch," and the premature roughening of bark on smooth-barked apple and pear trees were the types most commonly encountered. The latter type of injury was studied by Sorauer[38] about twenty-five years ago. By comparing Fig. 29, Plate XXIV (copied from Sorauer), with Fig. 2, Plate XXI, it is evident that the initial injuries were very similar in the two cases, except that that shown in Fig. 2 is much more severe. Sorauer found that this premature roughening is of especially frequent occurrence on rapidly growing varieties of fruit trees when they are from six to eight years old. The same cold spell that resulted in the bark-roughening described by Sorauer had also caused the bark of some trees to rupture and of others to "scald" or die to the wood in long patches. Some cambium, medullary rays, protoxylem, and pith tissues were killed and discolored; in the cortex the larger patches of dead collenchyma cells were subsequently cut off by new phellogen.

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[36] Lepeschkin, W. W., Zur Kenntnis der Einwirkung supramaximaler Temperaturen auf die Pflanze, Bericht. Deutsch. Bot. Ges. 30: 703. 1913.

[37] Overton, J. B., Studies on the Relation of the Living Cells to Transpiration and Sap-flow in Cyperus, Bot. Gaz., 51: 28; 102. 1911.

This type of bark and twig injury of pear trees was apparently also studied histologically by Miczynski.[39] He shows the distribution of dead and discolored tissues in a colored plate. In cases in which the cambium had been killed, the new cambium developed in the inner phloem much like that described in a former section of this paper. Another good but general account of bark injuries of fruit trees is given by Oberdieck.[40] He gives many clear details regarding numerous cases.

Sun-scorch is usually confined to trees that have not yet reached the rough-bark age, and consist of dead and discolored bark on the trunk or main branches, usually (though not always) on the west or southwest sides. Histologically its early stages are similar to those giving rise to the premature bark-roughening described by Sorauer. In many cases of the sun-scorch type, however, only the outermost collenchyma cells are involved, and consequently the resulting new bark surface looks only slightly frayed. Numerous interesting observations have been made on this bark disease, and in many of the discussions one may find pertinent suggestions. Hess,[41] for instance, notes that this trouble develops on smooth-barked forest trees one or more years after the forest has been thinned. Hartig[42] gives some interesting data along this line and concludes that the injury results from the contraction and expansion of the bark rather than from the heating of the sun as is maintained by many, because he found frequent cases of it on north slopes and on the east and north sides of trunks.

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[38] Sorauer, P., Über Frostschorf an Apfel- und Birnstämmen, Zeit. Pflanzenkr 1: 137. 1891.

[39] Miczynski, K., Ueber das Erfrieren der Gewebe des Birnbaums, Bot. Centralbl. 48: 228. 1891.

[40] Oberdieck, J. G. C., Beobachtungen über Erfrieren vieler Gewächse und namentlich unserer Obstbäume in kalten Wintern; nebst Erörterung der Mittel durch welche Frostschaden möglichst verhütet werden kann, pp. 108. Ravensburg. 1872.

[41] Hess, R., Der Forstschutz. Leipzig. 1878.

Many of the sections used in the present histological study of crown-rot were made from the bases of shoots arising from large branches of apple trees that had been pruned rather severely, and they therefore also represent the initial injuries preceding the development of crotch cankers as more fully discussed on pages 40–42 of my paper written in 1912. Goethe published a paper in 1877, in which he announced the conclusion that cankers are due to low-temperature injury of the bark. When it was pointed out to him that in Italy where the winters are mild cankers are equally prevalent, he reinvestigated[43] the matter and revised his conclusions to the effect that many of the cankers are due to fungus parasites. It should be noted, however, that his revised conclusion was based largely on the fact that in the spring of 1878 he found new cankers even though no *late* frosts had occurred. (The notion that only late frosts cause these injuries has led many astray.) Fungi developed on cankers when placed in moist chambers, but when spores were used on uninjured bark no cankers resulted. In the following winter bark injuries were numerous in crotches and other places where cankers usually occur. Many of the wounds were carefully cut out in April and most of them healed rapidly, although in a few instances the branches involved died.

Some years later, also, Goethe[44] made an extended study of winter-injuries, giving particular attention to the aftermath or the results of such injuries. A drop in mid October to − 2.5° C. and one to − 10° C. in November very severely injured the pith and other tissues in shoots and the bark of trunks just above the ground. High-headed trees were found more subject to trunk injury than low-headed ones. This is in agreement with what I found in western New York (Tech. Bull. 23, pp. 18–20). Goethe described interesting cases in which the different buds and nodes on the same branch varied greatly in their susceptibility to injury; some remaining normal while others were entirely killed.

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[42] Hartig, R., Ueber den Sonnebrand oder die Sonnenrisse der Waldbäume, Untersuch. Forstbot. Inst. München 1: 141. 1880.

[43] Goethe, R., Mittheilungen über den Krebs der Apfelbäume. Leipzig. 1877.

——, Weitere Mittheilungen über den Krebs der Apfelbäume, Landw. Jahrb. 9: 837. 1880.

[44] Goethe, R., Die Frostschäden der Obstbäume und ihre Verhütung, Nach den Erfahrungen des Winters 1879–80, dargestellt. Berlin. 1883.

Müller-Diemits and Stormer-Halle[45] found that fruit trees are most subject to bark diseases at the age when they first become profitable. The bark at the crown, crotches, and various other places on trunks and branches, according to these authors, dies; fungi and bacteria enter the wounds and induce further injury and decay. The wood becomes discolored, and the branch or tree involved dies.

As an illustration of the especial susceptibility of trees to bark injuries and the resulting diseases, during certain stages in their life history, especial attention may be called to the bark-roughening discussed above as well as to this paper by Müller-Diemitz and Stormer-Halle. The older literature of forestry contains many items of interest in this connection. Graebner,[46] for example, described a case of this kind, and in the writings of Hartig, Nördlinger, Hess, and others, are to be found many further instances. Graebner found that a high percentage of trees in a spruce forest had sustained bark injury on their trunks. Very many of them died of crown-rot. The trees had apparently been from 34 to 57 years old at the time the injuries occurred. In an adjoining spruce forest, where the trees were under 20 years of age, no bark injury could be found. It appears from various published statements that spruce trees in a forest stand usually become rough-barked between the ages of 30 and 40, depending upon the rate of growth and thickness of stand.

CONCLUSIONS

The histological study here briefly reported, in connection with my two former papers, throws enough light on the earlier stages of crown-rot to permit more definite and general statements regarding its development. It is shown that this and some related bark diseases are not due primarily to the organisms usually found in such affected bark in summer, but to injuries arising when adverse environmental conditions overtake trees having immature bark in certain regions. The rotting of the dead or dying bark is due chiefly to fungi which in

[45] Müller-Diemitz, J., und Stormer-Halle, K., Das Obsthaumsterben, Deutsch. Obstbauzeit. 56: 81. 1910.

[46] Graebner, P., Beiträge zur Kenntnis nichtparasitärer Pflanzenkrankheiten an forstlichen Gewächsen, Zeit. Forst. Jagdwesen. 38: 705. 1906.

some cases also kill living portions while vegetating in severely injured bark.

On Plate XXI are shown some of the main types of injuries often found in bark after unseasonably severe periods. This material was collected before evident growth started in the spring and therefore gives some idea of the actual distribution of the injuries. An examination of these figures makes it appear that injuries are of two types: in Figs. 1, 2, 4 and 8 they are evidenced chiefly by a discoloration and collapse of the affected tissues, whereas in Fig. 3 the injury consists mainly of a tangential rupture with only a few of the groups of discolored cells; in Figs. 5, 6, 7 and 9 there occur combinations of the two types of injury. In the latter cases the tissues along the margins of the ruptures are discolored and collapsed much as they are in Figs. 1 and 2. Unfortunately the sections of the material having a combination of the types of injuries shown in Figs. 1 and 2, or of those in Figs. 2 and 7, turned out to be such poor preparations that no use could be made of them.

Plate XXII shows comparable cases as they appeared about two weeks later. This represents a stage of regeneration growth during which living parenchyma cells surrounding injured or dead regions are actively proliferating into spaces formerly filled by the shriveling masses, and into gaps occasioned by ruptures. Figs. 10 and 12 are especially interesting because dead tissues are compressed into more or less radially arranged plates. The proliferating cells are seen to penetrate many of the dead masses, and apparently make contact with living cells beyond. This rapid early regeneration-growth in injured bark is responsible for the fact that so few injured places result in patches of dead bark.

Practically the final alignment of injured and living tissues, as well as the locations of the new meristematic layers, is shown on Plates XXIII and XXIV. From these figures it is evident that when enough of the cambium and inner phloem are killed to form a fairly thick dead layer only a few, or in some cases over considerable areas no living connections are re-established between the old wood and the bark. In some instances the most severely affected bark died early (Figs. 24, 25 and 26), while in others (Figs. 18, 21, 22, 28, 31 and 32) regeneration went on rapidly and the formation of a considerable amount of new wood and bark resulted (Figs. 18, 22 and 31). There are some in which the outer bark has remained alive but in which very little effective regeneration has resulted (Figs. 23, 30, 32 and 35). In most of the last type of cases the bark died to the margin of the severe injury before mid-summer, so that they appeared like Figs. 24 and 26. In instances like those of Figs. 21, 30 and 31 the results were sometimes most striking, because often a fair beginning had been made on the new growth of wood when suddenly the bark died over large areas. In case such an injury occurred on a small branch or shoot, it usually died outright as shown in Fig. 39*a*.

The transition from stages like those shown in Figs. 23, 32 and 35 to those of Figs. 24, 25, 26, 41, 46, 47 and 51 seems fairly clear. The associated micro-organisms evidently play an important rôle in the decay or disintegration marking the later stages of these bark diseases, and in some types doubtless extend the injured areas by their vegetative activities in the places initially killed (Figs. 27, 30, 33 and 35). Yet, this does not seem to be generally the case. The tree shown in Fig. 50 had lived at least fourteen years after the occurrence of the injury, eventually resulting in "heart rot." It is evident from this figure that the fungus rotting the wood present at the time the initial injury occurred has not progressed far outside the last layers of injured wood. In fact, it appears as though it may have rotted only as much of the wood formed since the occurrence of the injury as had been discolored by the diffusion of disintegration products from the initially killed cells. This diffusion injury is shown in Figs. 39 and 42. Yet, in looking over the figures of Plate XXIV it becomes obvious that once a wound parasite, or even a saprophyte especially adapted to a particular host, gains entrance to such an admixture of dead and regenerated living tissues, some living portions may be killed as a result of the vegetative activities of the fungus.

This record of low-temperature injuries occurring in the bark of fruit trees, and of their subsequent development into bark diseases, is of interest and value independent of the factors that give rise to the initial injuries. The diseases in question are thus traced so much nearer to their first causes. Both the macroscopic and the microscopic appearances of much of the bark affected indicate that excessive tensions are developed during the occurrence of the injuries. It remains to be determined whether or not the tension-injury hypothesis of Sorauer is applicable to low-temperature injury in general, in connection with the physiological disturbances induced by the occurrence of severe weather while some of the bark tissues are in certain stages of immaturity or arrested development. It seems possible that, at least in some cases, the presence in the bark of metabolized foods of insufficient concentration to allow normal growth and maturation is the most significant phase of immaturity; the occurrence of droughts appears to have a significant relation. An adverse period in the environment occurring at such a time stops the further accumulation of the labile components of protoplasm, and a long retention of these elementary constituents, together with the enzymes present, may lead to catabolic processes that eventually result in the death of the tissues involved.

ACKNOWLEDGMENTS

In this connection it is a pleasure to acknowledge the aid received from Professors J. B. Overton, L. R. Jones, and E. M. Gilbert, of the University of Wisconsin, during the winter of 1911–12, and especially that of Professor C. E. Allen, of the same institution, for the many excellent suggestions he made regarding the revision of this paper.

DESCRIPTION OF PLATES XXI–XXVII

PLATE XXI

Sections of material collected before growth started in the spring, showing types of initial injuries. All from apple excepting Figs. 1 and 2, which are from pear.

FIG. 1. Injury mainly in cambium, phellogen, and phloem regions; scattered cell-groups in xylem and cortex affected, as indicated by discolored places.

FIG. 2. Another section with severe initial injuries confined chiefly to inner cortex and outer phloem; phloem rays and cambial zone also injured. Scattered groups of dead cells in other parts of cortex and phloem.

FIG. 3. Section of apple branch with common type of injury not usually accompanied by much discoloration, consisting principally of a rupture in the inner phloem and only secondarily of groups of dead, discolored cells.

FIG. 4. Section of apple with most of the injury in the inner phloem, as indicated by discolored streaks and masses. On the left the cambium is killed; on the right it is alive and apparently normal.

FIG. 5. A condition comparable with that of Fig. 4, excepting that a portion of the injury consists of ruptures as shown near the right.

FIG. 6. Some living cambium on the right, and a zone of severe injury in the inner phloem above; on the left most of the cambium is killed. The rays are more severely affected than in the section shown in Fig. 5.

FIG. 7. Collapsed tissue in the inner phloem and cambium is interspersed with a few living cells. Phloem rays are dead, and scattered groups of dead cells occur in the older portions of the phloem.

FIG. 8. Some groups of dead cells and some ruptures occur in the cambium; the inner phloem has but few affected cells. The middle and outer phloem are considerably injured.

FIG. 9. Much like Fig. 7, but with larger groups of living cells in the cambium; more definite radial ruptures are evident in the inner phloem. On the right all of the phloem and cambium between the clefts and the wood are dead.

PLATE XXII

Displacement of initially killed tissues by regeneration-growth. Collected two weeks after the material used for Plate XXI; all from apple.

FIG. 10. A development from an injury like that shown in Figs. 6 and 7. All living cells have grown and compressed the dead material. Living cells proliferate outgrowths into the dead masses. Regeneration tissue is all parenchymatous; it divides in all planes.

FIG. 11. Similar to Fig. 10; in neither of these instances are living connections evident between the bark and wood.

FIG. 12. Different from the two preceding chiefly because some of the cambium has survived. Even where the cambium is killed, frequent living cells are found to bridge the gap. Regeneration has repaired many holes that resulted from the shrinking of groups of dead cells.

FIG. 13. Seems to be a later stage of an initial injury of the type show in Fig. 6; both at the right, where the cambium is involved, and at the left, where the inner phloem is affected, proliferating cells connect the wood with the bark through mantle of dead tissue.

FIG. 14. Though the old cambium (*oc*) was only slightly injured, a new one has begun to develop (*cc*) in the regeneration tissue of the inner phloem. Initial injury in the older phloem was very severe, yet the living portions of it are compressing the dead masses by growth.

FIG. 15. From the type shown in Fig. 8; new cambium (*cc*) is forming in regenerated tissue of the inner phloem, and the former cambial line is discolored. The outer phloem and cortex are almost wholly isolated from the inner bark by a ragged layer of dead tissue. Occasional living cells occur in the dead mass.

FIG. 16. Derived from a portion that was less injured than that shown in Fig. 6. Living tissues dominate, and new cambium (*cc*) has become established.

FIG. 17. Much like Fig. 15, excepting that the injury is much more severe in the old cambium.

PLATE XXIII

Final disposition of much of the initially killed tissue. Collected a month after that shown in Plate XXII; all from apple.

FIG. 18. Late stage of a type shown in Figs. 7 and 9. Former position of the cambium (*oc*) and its present location (*nc*). A considerable layer of new wood (*nw*) has developed, and a new phloem (*np*) is also noticeable. In the phloem the dead masses are localized near the boundary between the old and the new phloem, some extending into the new phloem toward the cambium just as dead streaks extend into the new wood from the injury outside of the old wood (*ow*).

FIG. 19. From the middle toward the right of the figure the new cambium (*nc*) is only faintly indicated. Injury at the old cambium (*oc*) is not as conspicuous as in Fig. 18, but it is of wider extent. Initial injury occurred throughout the old phloem and was very severe.

FIG. 20. Mantle of greatest injury with irregular course. At the right the new cambium (*nc*) has begun the development of a new layer of wood (*nw*), while at the left no substitute cambium is yet visible.

FIG. 21. More irregularity in the course of the mantle of greatest initial injury. Living phloem is left attached to the old wood (*ow*) and converted into wood without leaving an active cambium. New cambium (*nc*) has developed outside the zone of injury that produced the layer of wood. The cambium has become abnormal, yellowish in color, and is partly disorganized. Discolored streaks extend from it into both new wood (*nw*) and new phloem (*np*).

FIG. 22. Much like Fig. 18, showing in addition a new phellogen (*ph*) cutting off the outer part of the cortex.

FIG. 23. Exaggerated form shown near the left end of Fig. 20. No new cambium is in evidence, and dead matter predominates, although from the outside the bark appeared normal.

FIG. 24. A case in which the most severely injured portions of the bark died and a callus (*cal*) developed along its margin. New wood (*nw*) at the lower right arose without leaving cambium.

FIG. 25. Severe initial injury confined to a small space. Although much isolated from the old wood, the callus is normal, having an active cambium. As in Fig. 24, fungus mycelium is present in the dead bark and in the dead mantle between the callus and the dead wood.

FIG. 26. Bark half-way around stem is dead and sunken, much like the patch shown in Fig. 41. In cross-section this looked like a miniature of the specimen shown in Fig. 50. Mycelium and pycnidia of a bark fungus were present.

PLATE XXIV

Higher power views of some injured tissues in the stage shown on Plate XXIII.

FIG. 27. View of a region like that in *oc* of Fig. 18. Living connections through the dead region are evidently few and imperfect.

FIG. 28. Like Fig. 27. Former phloem rays have become discontinued and have undergone division and become converted into callus tissue.

FIG. 29. Copied from Sorauer's paper on "Frostschorf" of apple and pear in *Zeitschrift für Pflanzenkrankheiten*, 1: 137–45. 1891. Cortical injury that usually precedes premature bark-roughening.

FIG. 30. Magnified view of the type shown in the center and right of Fig. 19; substitute cambium (*nc*) developed in a meandering course. Much-injured bark practically isolated from the old wood (*ow*).

FIG. 31. Detail of a case something like that shown in Fig. 22, excepting that practically no new phloem has yet developed; new cambium (*nc*) is considerably disorganized and discolored. Old phloem (*op*) is permeated by initially killed tissue, in direct contact with disintegrating new cambium.

FIG. 32. Much like Fig. 30; only a faint indication of substitute cambium (*nc*) is in evidence. Injury in old phloem is more severe than in Fig. 30; cells in regeneration-growth are less affected by pressure than those in Fig. 30.

FIG. 33. Higher power view of case like that in left-hand portion of Fig. 20, with uncommonly thick mantle of dead tissue. Living portions of former bark rays are converted into ordinary parenchyma.

FIG. 34. Cross-section of a large dead streak of phloem surrounded by modified irregular parenchyma.

FIG. 35. Similar view of a living streak in the phloem surrounded by layers of dead, collapsed cells.

PLATE XXV

Initial injuries followed by another type of regeneration. All from maple except Fig. 41, which is from pear.

FIG. 36. Series of magnified views of a portion of Fig. 37, from old bark (*ob*) to old wood (*ow*): *a*, old bark (*ob*) run through by rifts, new bark (*nb* 1) with inclusions of dead masses, and new cambium (*nc* 1) just outside some new wood shown at the outer edge of outer new wood (*nw* 1) in Fig. 37; *b*, from the inner edge of the outer new wood (*nw* 1) of Fig. 37, showing the new cambium (*nc* 2) and some very irregular new bark (*nb* 2); *d*, from the outer edge of the inner sheath of new wood (*nw* 2) of Fig. 37, *nb* 2 and *nb* 3 together constituting the compressed new bark between *nw* 1 and *nw* 2 of Fig. 37. *e*, higher power view of line *oc* of Fig. 37, showing some detail.

FIG. 37. Cross-section of maple tree (*Acer platanoides*) with a season's growth added after the occurrence of the initial injury, that had been similar to that shown in Fig. 3, and somewhat like that shown in Fig. 38. Three cambial layers have developed in place of one. The tangential cleft left some living phloem adhering to the old wood like that shown in Fig. 3. Substitute cambium arose in the strip of inner phloem adhering to the wood, giving rise to *c* 3 of Fig. 37; then along the inner surface of the loosened outer bark another cambium developed which gave rise to new wood in its *middle* and was thus divided into two cambial sheaths (*c* 1 and *c* 2), each producing wood and bark. Activity of three cambial layers, as detailed in Fig. 36, gives rise to unsightly enlargements like that shown in Fig. 40.

FIG. 38. Section of box-elder tree (*Acer Negundo*) with portion of its bark separated from the wood, though still living. Beginning of callus formation is shown along the edges of the loose bark (May 28).

FIG. 39. Higher power view of a portion shown in Fig. 26*e*; considerable regeneration-growth of wood outside the zone of initial injury, which subsequently died and became discolored.

FIG. 40. Trunk of a street tree (*Acer platanoides*) unduly enlarged near the upper part of the trunk owing to the activity of three cambial zones developed after the occurrence of some injuries initially much like those shown in Fig. 3.

FIG. 41. Trunk of a smooth-barked pear tree in early summer, with a sunken patch over the places sustaining most severe internal injuries.

FIG. 42. Detail view of a section taken across the faintest portion of the line *oc* in Fig. 37, showing that normal new wood (*nw*), arising outside such a line of initial injury, may subsequently be killed and discolored.

PLATE XXVI

Collection of bark-injured and crown-rotted stems, in which the injury was accompanied by radial clefts. All of apple except Fig. 48, which is of orange.

FIG. 43. Apple tree with nearly complete girdle of loose bark (one patch opposite) and a radial cleft 17 cm. long.

FIG. 44. Shows the extent of the loose bark of Fig. 43.

FIG. 45. Twelve-year-old apple tree which had a complete girdle of loose bark from the ground up to the main branches. A radial cleft 25 cm. long occurred in it near ground.

FIG. 46. Apple tree with complete girdle of dead bark; thick callus along its upper edge.

FIG. 47. A typical case of crown-rot on apple.

FIG. 48. Stem of orange tree showing radial clefts in loose bark. Initial injury occurred on the night of November 20, 1914, when the temperature sank to a little below − 2° C. In the summer of 1915 many trees affected in this manner died with symptoms of "withertip."

PLATE XXVII

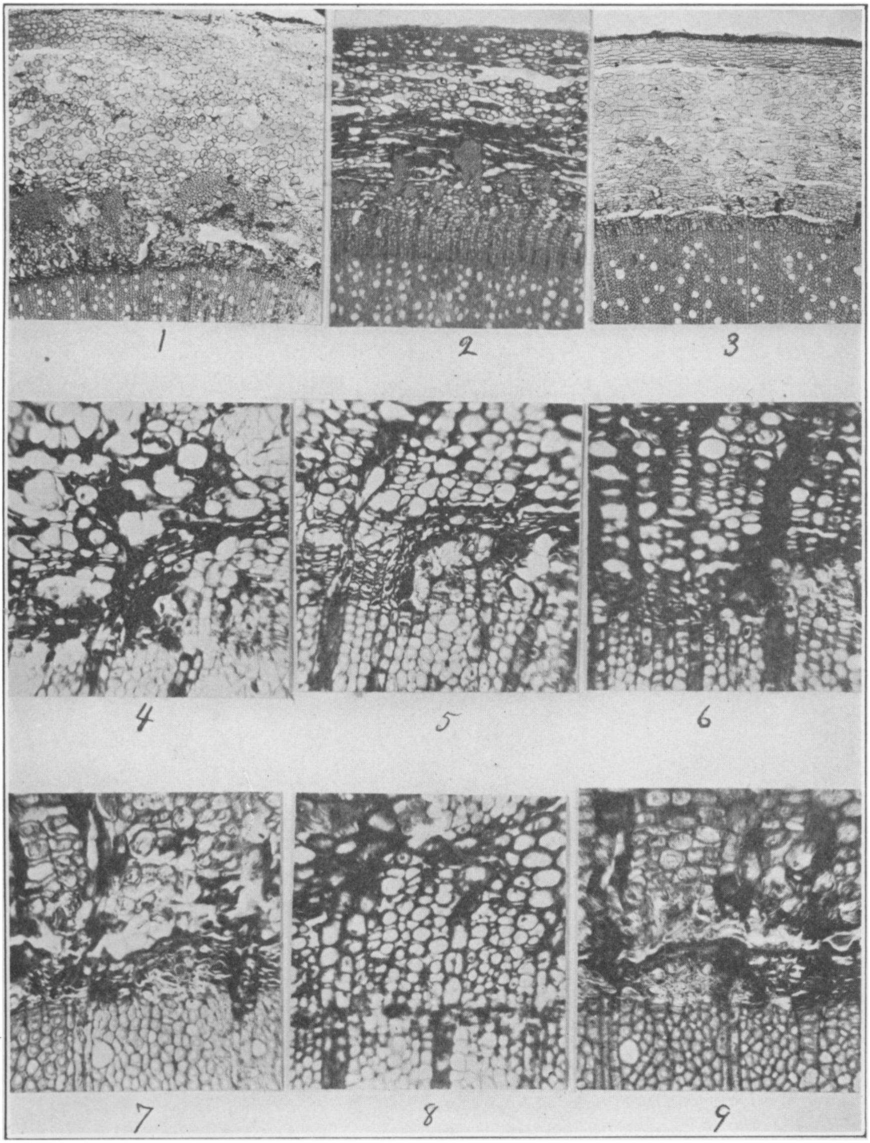
Crown-rot and other troubles of large trees.

FIG. 49. Section from near the base of a large apple-tree trunk (28 cm. in diameter), showing a line of initial injury that occurred some fourteen years before cutting; also showing that the bark sustained a radial cleft (upper side).

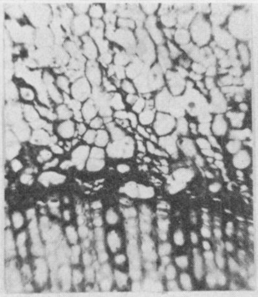
FIG. 50. Section of another tree of the same size and from the same orchard as that shown in Fig. 49. The initial injury occurred in the same year as that in Fig. 49. The wood cylinder subsequently died and rotted, and some of the wood produced by the new cambium also decayed.

FIG. 51. Large apple tree with complete crown-rot girdle. Upper roots died, but those under the center of the tree were alive.

FIG. 52. Section of a spruce stem, copied from Hartig (*Untersuch. Forstbot. Inst. München* 1: 147. 1880). Included here to show that the initial injury from which the trouble developed occurred during the dormant season and not during the growing season as was maintained by Hartig.



GROSSENBACHER: CROWN-ROT OF FRUIT TREES.



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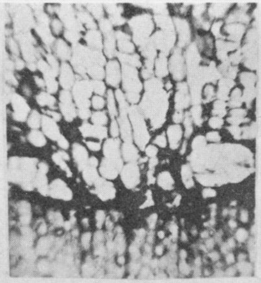

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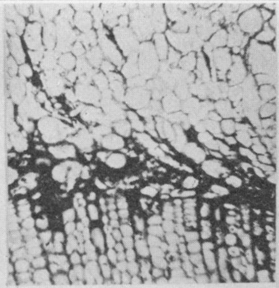

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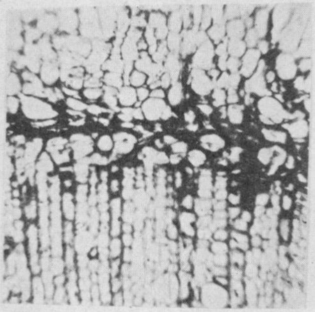

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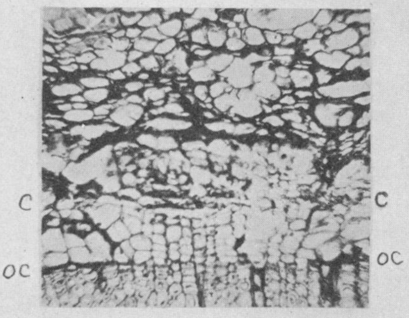

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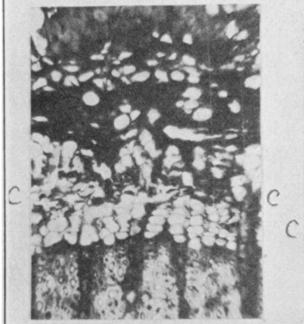

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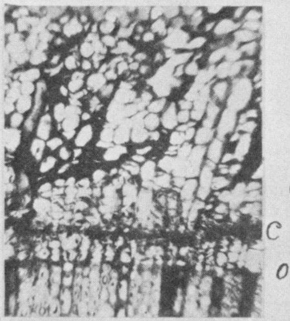

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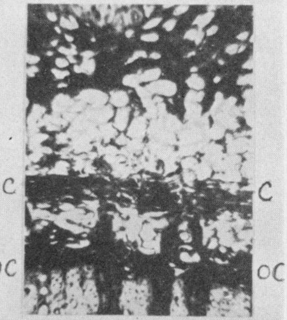

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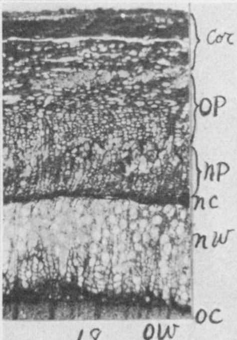
GROSSENBACHER: CROWN-ROT OF FRUIT TREES.



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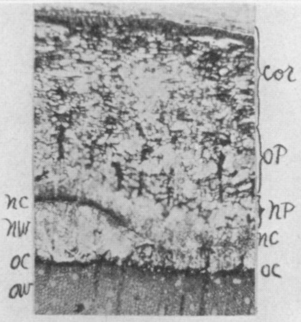

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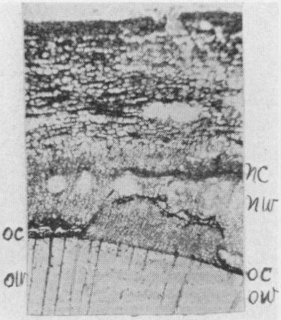

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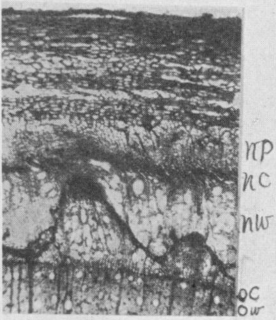

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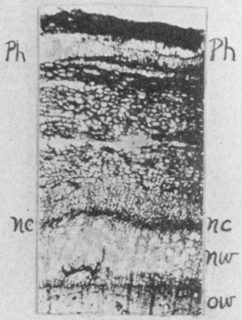

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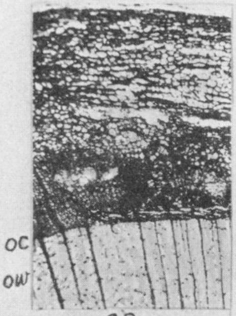

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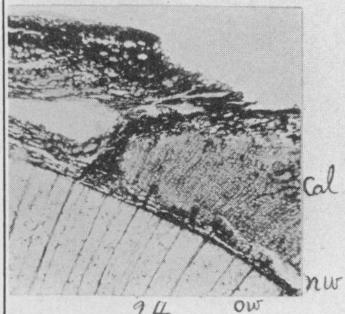

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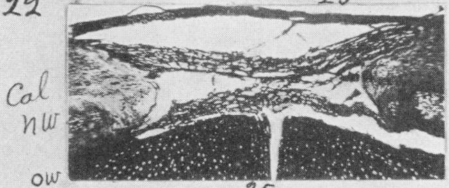

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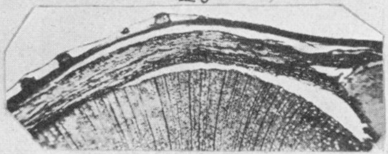

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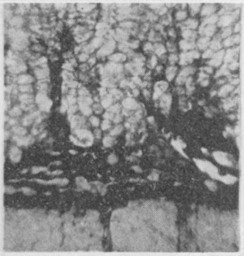
GROSSENBACHER: CROWN-ROT OF FRUIT TREES.



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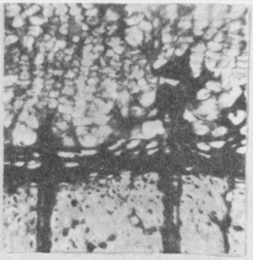

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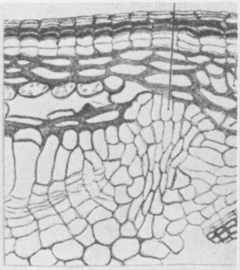

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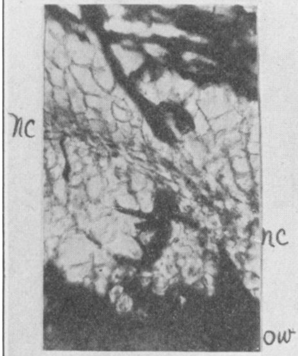

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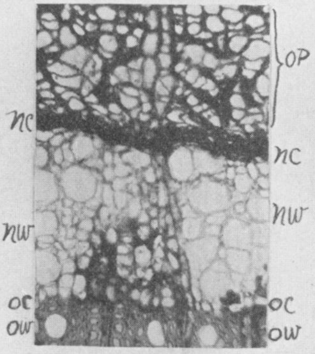

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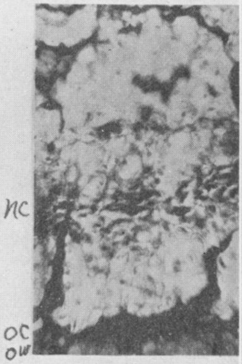

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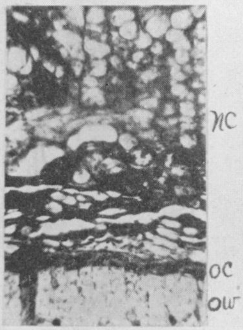

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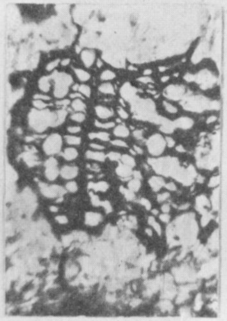

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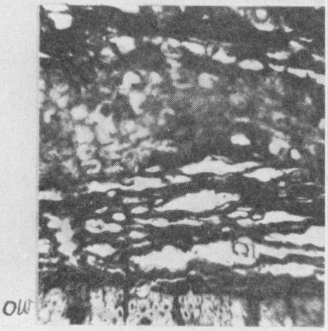

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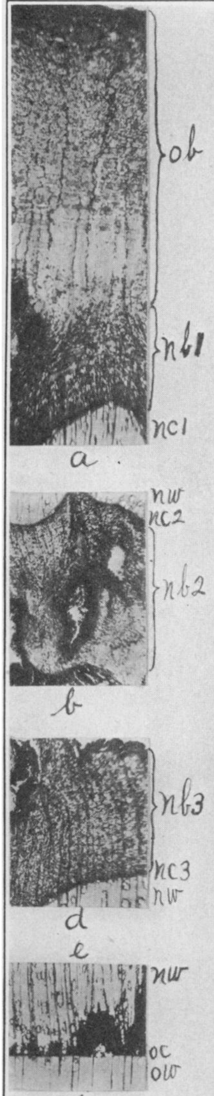
GROSSENBACHER: CROWN-ROT OF FRUIT TREES.



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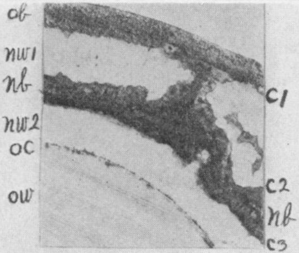

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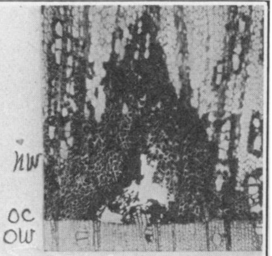

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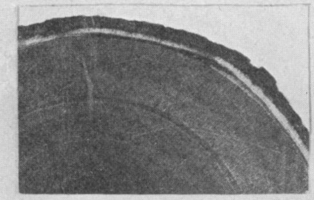

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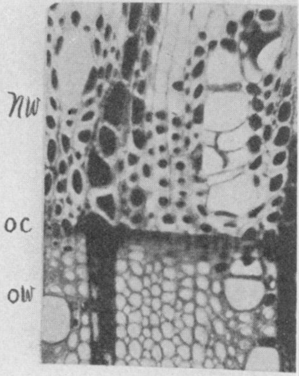
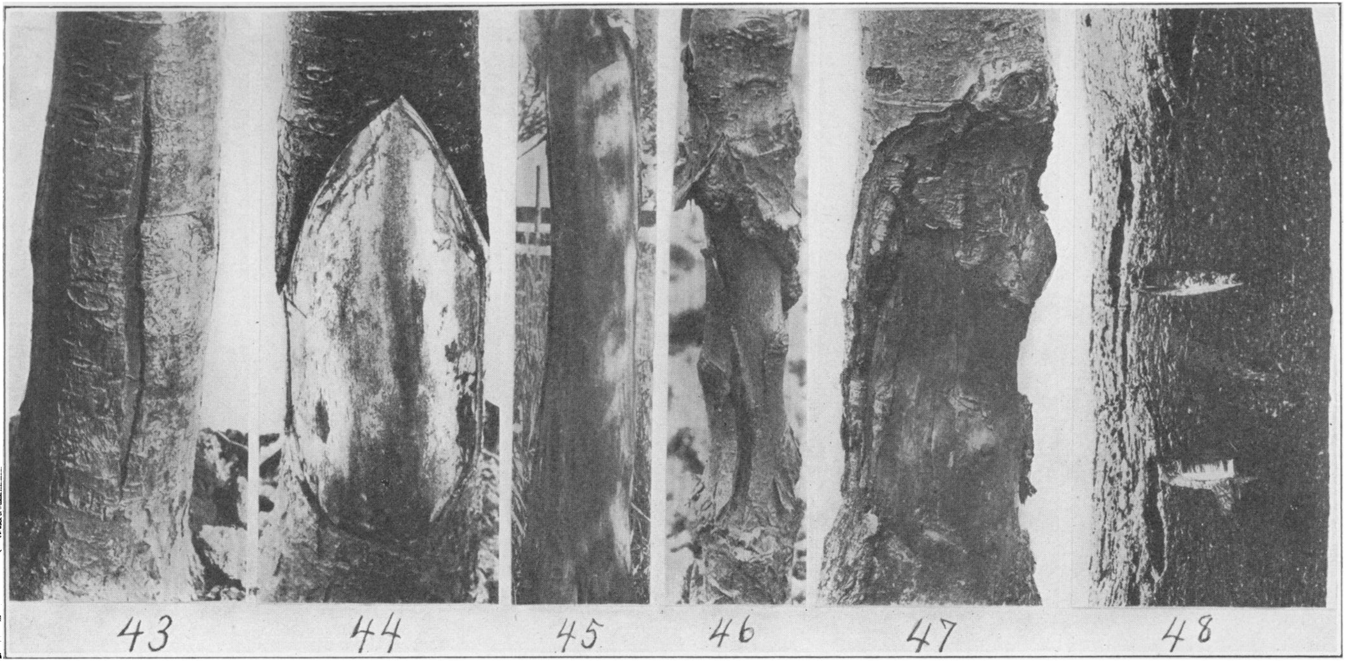
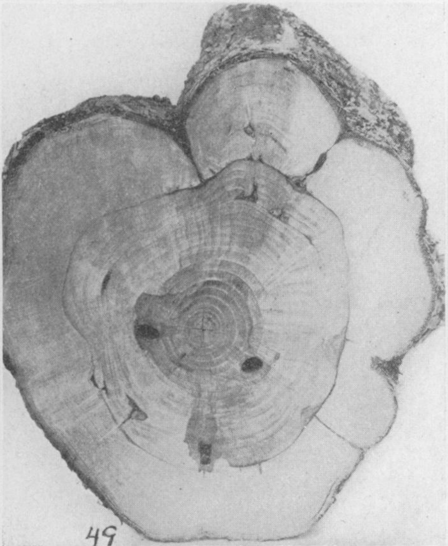


GROSSENBACHER: CROWN-ROT OF FRUIT TREES.



GROSSENBACHER: CROWN-ROT OF FRUIT TREES.



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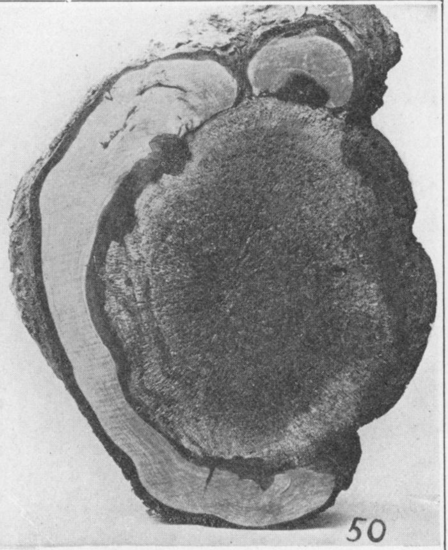

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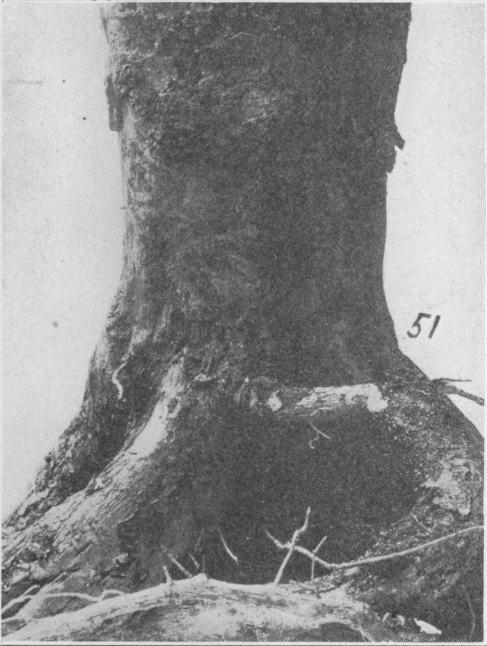

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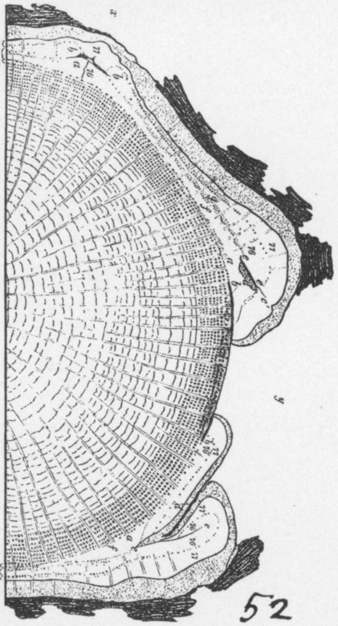

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GROSSENBACHER: CROWN-ROT OF FRUIT TREES.